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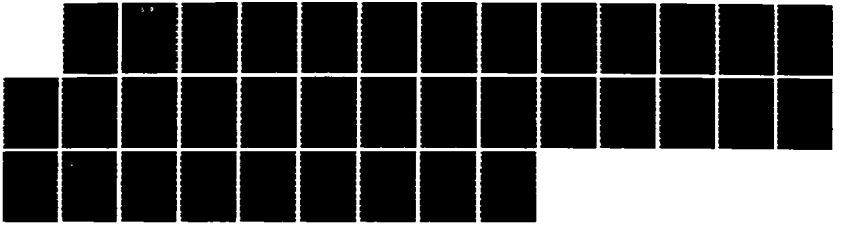

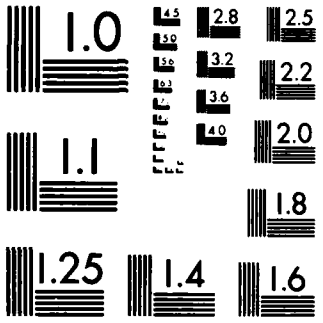

OPERATION EVEREST II: Preservation of cardiac function at extreme altitude

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Hypoxia at high altitude could depress cardiac function and decrease exercise capacity. If so, impaired cardiac function should occur with the extreme, chronic hypoxemia of the 40-day simulated climb of Mt. Everest (8840m, P_B 240 mmHg, PIO_2 43 mmHg). In the five subjects having resting and exercise measurements at the barometric pressures of 760 (sea level), 347 (6100 m), 282 (7620m), and 240 mmHg, heart rate for a given oxygen uptake rose with the progressive hypoxia. Slight (6 beats/min) slowing of the heart rate occurred only during exercise, with arterial blood oxygen saturations below 50%. Oxygen breathing reversed hypoxemia but never increased heart rate, suggesting that depression of rate, if present, was slight. For a given oxygen uptake, cardiac output was maintained. The decrease in stroke volume appeared to reflect decreased ventricular filling i.e., decreased right atrial and wedge pressures. Oxygen breathing did not increase stroke volume for a given filling pressure. We concluded that extreme, chronic hypoxemia caused little or no impairment of cardiac rate and pump functions.

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Abstract

Hypoxia at high altitude could depress cardiac function and decrease exercise capacity. If so, impaired cardiac function should occur with the extreme, chronic hypoxemia of the 40 day simulated climb of Mt. Everest (8840m, P_B 240 mmHg, PIO_2 43 mmHg). In the 5 subjects having resting and exercise measurements at the barometric pressures of 760 (sea level), 347 (6100m), 282 (7620m), and 240 mmHg, heart rate for a given oxygen uptake rose with the progressive hypoxia. Slight (6 beats/min) slowing of the heart rate occurred only during exercise, with arterial blood oxygen saturations below 50%. Oxygen breathing reversed hypoxemia but never increased heart rate, suggesting that depression of rate, if present, was slight. For a given oxygen uptake, cardiac output was maintained. The decrease in stroke volume appeared to reflect decreased ventricular filling i.e., decreased right atrial and wedge pressures. Oxygen breathing did not increase stroke volume for a given filling pressure. We concluded that extreme, chronic hypoxemia caused little or no impairment of cardiac rate and pump functions.

Introduction

At high altitude, the capacity for exercise decreases (3,5-8,10,14,19,24,25), probably, in large measure, because of hypoxemia. At extreme altitude, the hypoxemia becomes extreme (11,16,18,22,24). Severe hypoxemia could depress both the heart rate (3,6,18,20) and myocardial contractility (2,12,23), thereby contributing to the decreased exercise capacity at high altitude. But if factors other than cardiac function depress maximum oxygen uptake, the cardiac response could be appropriate for the oxygen utilized. We considered that hemodynamic measurements at cardiac catheterization should provide clues to separate these possibilities, and indicate whether or not the human heart functions normally at, or near the limits of tolerance to chronic hypoxia. Such information would be useful in understanding the capacity for adaptation to hypoxia of the normal human heart and for providing insight into changes expected in disease states.

Methods

Our strategy was to measure rest and exercise stroke volumes and heart rates, during a simulated climb of Mt. Everest. If there were hypoxic depression of the heart rate, then one might expect: a) depressed heart rates for a given oxygen uptake, b) failure of the normal increase in heart rate

during exercise, and c) increased heart rates with removal hypoxia. If marked hypoxic depression of stroke volume occurred, then a decrease in stroke volume at a given ventricular filling pressure should be seen, and the decrease might be reversed by oxygen breathing. As previously described, the use of the altitude chamber in this multidisciplinary study allowed the subjects to be decompressed during 6 weeks of acclimatization where measurements were made with relative safety and good environmental control. The exercise intensity was measured in terms of both physical work and oxygen uptake. Oxygen uptakes at submaximal work loads were compared to maximum uptake reported by Cymerman et al. (7) measured at each barometric pressure.

The subjects were 8 healthy volunteers age 21 to 31 years. Their physical characteristics, details of the study protocol and the cardiac catheterization procedure, and the description of the altitude chamber, are in other reports (7,11,16,22). For the purposes of this report, variables measured at rest and for each exercise included continuous recording (Gould 8 channel recorder, model 200) of heart rate from a precordial lead, oxygen saturation from an ear oximeter (Hewlett Packard, model 47201-A), minute ventilation from a dry gas meter (Parkinson Cowan, model CD-4), mixed expired oxygen and carbon dioxide concentrations

(Perkin Elmer Mass Spectrometer, models 100 A and B), and intravascular pressures (right atrial, pulmonary arterial, pulmonary arterial wedge, and systemic arterial) using Statham pressure transducers. For pressure measurement, zero was set 5 cm below the sternal angle with the subject sitting on the cycle ergometer and in his preferred cycling position. Throughout the procedure we checked to see that the position of the thorax had not changed relative to the transducer, because such change would introduce error into the pressure measurements. The error would be relatively greater for pressures of small absolute value, i.e., right atrial and wedge pressures. Systemic arterial blood samples were placed on ice and analysed immediately for pH, PO_2, PCO_2, and oxygen saturation using the Instrumentation Laboratories Co-Oximeter (model 282). The comparison between the ear oximeter reading and that of arterial blood drawn simultaneously, Figure 1, indicated that above approximately 35% by ear oximeter, the two saturations were linearly related. At rest and for most exercise levels, cardiac output measured by three methods showed good agreement (11). Thermodilution outputs are reported, but where not available, we have utilized the Fick methods employing oxygen or multiple inert gases.

The measurements in the text, tables and figures are presented as mean $\pm$ one standard error of the mean (SEM).

Comparisons between pairs is by paired t test, and between groups is by two-way analysis of variance for equal sample sizes. Relationships between two variables was by linear regression. For sequential exercise levels of increasing intensity, the relation between heart rate and oxygen uptake was assessed by linear regression for each individual. The resultant slopes and y intercepts were averaged to obtain the average linear regression for the group. Differences between groups or correlation coefficients were considered significant when $p < 0.05$.

Results

Individuals, reported by exercise intensity, Tables 1, 2, and 3, had similar relative exertions as indicated by oxygen uptake as a per cent of maximum. Heart rates, for an absolute oxygen uptake were higher at reduced barometric pressures (P_B) than at sea level, Table 4, Figure 2. In 5 subjects where the heart rate during an exercise was examined as barometric pressure fell, Figure 3, rest and exercise heart rates progressively rose. Heart rate rose rapidly during the first 30 sec of exercise at all barometric pressures, Figure 3. At P_B 760, 347, and 282 mmHg, the average heart rates at 2 min of exercise were equal to, or higher than, the 30 sec value. However, at P_B 240, the 30 sec measurement represented the maximal heart

rate, and by 2 min the rate had fallen in all 5 subjects (mean change, -6 beats/min).

Arterial oxygen saturations remained high during exercise at sea level. With increasing altitude resting saturation progressively decreased. Saturations fell further with exercise, Figure 3. At 30 sec of exercise at P_B 240, the time of the peak heart rate, the saturations by ear oximetry averaged 45%, Figure 3, and when the heart rate had fallen at 2 min, they averaged 40% (equivalent to directly measured saturation of 50%, and PaO_2 of only 27 mmHg (22). Oxygen breathing decreased the resting heart rate at P_B 282 and 240 mmHg, and decreased the exercise heart rates at P_B 347 and 282, Table 5. One subject was given oxygen during exercise at P_B 240, and in him, oxygen did not increase heart rate.

To determine whether the heart rates during maximal exercise at high altitude were appropriate for the work which could be accomplished, we compared the heart rates at maximum oxygen uptakes as reported by Cymerman et al. (7), with those during submaximal exercise at sea level, Figure 4. There was a strong correlation between the maximum oxygen uptake and the simultaneously measured heart rate. At the reduced barometric pressures, the heart rates at maximum oxygen uptake exceeded the heart rates at sea level for a given oxygen uptake, Figure 4.

At all barometric pressures studied, cardiac output increased progressively as exercise intensity increased, Tables 1, 2, and 3. Also at all barometric pressures, cardiac output values, for a given oxygen uptake, were not less than those at sea level, Figure 5A. However, for a given heart rate, cardiac output values were lower at the reduced barometric pressures, Figure 5B.

Stroke volume at sea level increased sharply from rest to 60 watt exercise, but heavier exercise caused little further increase, Table 1, Figures 5C and 5D. At reduced barometric pressures, stroke volumes were less than at sea level, and did not plateau with increasing exercise. Compared to sea level values, high altitude right atrial pressures were decreased for 10 of the 15 resting and for 20 of 23 exercise measurements, Tables 1, 2, and 3. The mean right atrial pressures tended to be low despite the pulmonary hypertension. For the barometric pressures examined, stroke volumes were similar for a given right atrial pressure, Figure 6A. The resting wedge pressures at reduced barometric pressures were not altered from sea level values but all 18 exercise values were reduced, Tables 1, 2, and 3. Mean systemic arterial pressure was unchanged. At P_B 347 mmHg, stroke volume was not reduced for a given wedge pressure, Figure 6B. Oxygen breathing did not consistently increase

stroke volume for a given right atrial or wedge pressure, Figure 7.

Discussion

The main findings in the normal volunteers of the present study were that heart rate for a given oxygen uptake and stroke volume for a given right atrial or wedge pressure, were well preserved at simulated high altitude near the limits of human tolerance to chronic hypoxia. The heart rates were accurate, being taken directly from the recording paper. The thermodilution measurements of cardiac output agreed well with two independent measurements of cardiac output using the Fick method (11). The intravascular pressures were directly measured and the reference zero was repeatedly checked relative to the transducer and to the subject's position. The decreased wedge pressures we observed at high altitude were consistent with the decreased left ventricular volumes independently measured using 2 dimensional echocardiography (21) in these subjects. From the above, we considered that the main findings in the present study were based on reliable measurements of heart rate, cardiac output, and intravascular pressures.

At rest, the finding that the resting rate increased with increasing altitude, indicated net stimulatory effects of

altitude. Hypoxemia was the likely cause of the increase in rate because with increasing altitude, the hypoxemia became severe and the heart rate decreased when the hypoxemia was relieved by oxygen. During submaximal exercise, the prompt rise in heart rate and the finding of the highest rate at the highest altitude, was compatible with a normal rate function in the absence of hypoxic depression. Had there been marked hypoxic depression of the heart rate, one would have expected a rise in rate with oxygen breathing, whereas the rates fell. However, the small fall in heart rate near the end of exercise at the highest altitude suggested there may have been some rate depression. A similar observation was reported in one subject exercising at 5800 m (18). Both in this subject and in our subjects, a role for hypoxia was suggested in that the decrease in heart rate during exercise only occurred with severe hypoxia. Hypoxic depression of the heart rate, if present, was of modest degree, and occurred only with severe hypoxemia.

The fall in maximum oxygen uptake may be relatively independent of heart rate. At 4300m para-sympathetic blockade with atropine increased the heart rate during maximal exercise from 165 to 176 (13), and beta-adrenergic blockade with propranolol decreased rate from 178 to 135 (17), but neither maneuver altered maximal oxygen uptake. Maintained maximum oxygen uptake for such a wide range of

heart rates, suggests that rate may not be an important determinant of maximum oxygen uptake at 4300m, if compensatory changes in stroke volume can occur.

Usually, the increased heart rate at high altitude has been accompanied by a decreased stroke volume (1,2,12,18). Our measurements indicated that the decrease in stroke volume was offset by the increase in heart rate, even during exercise at the highest altitude, such that the cardiac output was maintained. Previous cardiac output measurements have not been made in man at barometric pressures equivalent to the summit of Mt. Everest, but measurements at lesser altitudes have usually indicated maintained (4,6,15,18) or somewhat reduced (2,12) cardiac output values for a given oxygen uptake.

The stroke volume could be reduced because of reduced cardiac filling or because of reduced contractility. Reduced filling pressures could result either from the tachycardia or from a reduction in blood volume, or both. Ultrasound studies of Suarez et al. (22) in the subjects of the present report, indicated a consistent reduction in left ventricular volumes at rest and during exercise. Our findings of reduced right atrial and wedge pressures implied that reduced ventricular filling was one possible cause of the reduced stroke volumes. That the stroke volume was not depressed for a given filling pressure suggested maintenance of

contractile function. That oxygen breathing did not increase stroke volume for a given filling pressure, suggested that there was not severe hypoxic depression of contractility. The increase in stroke volumes with exercise at high altitude indicated that resting stroke volumes were not maximal. These findings plus the normal relationship of cardiac output to oxygen uptake suggested that contractile cardiac function was maintained in chronic, severe hypoxia.

Independent support for the maintenance of contractile function in these subjects at high altitude came from the two dimensional echocardiographic study (21), where at the barometric pressure of 282 mmHg, compared to sea level, ejection fraction, the ratio of peak systolic pressure to end-systolic volume, and mean normalized systolic volume at rest were sustained. Indeed, during 60 watt exercise the ejection fraction was higher (79±2 vs 69±8 %) at P_B 282 than at sea level. We conclude, as did Suarez et al (21), that despite the decreased cardiac volumes, the hypoxemia, and the pulmonary hypertension, cardiac function appeared to be maintained. The implication was that depressed cardiac function was not responsible for reduced exercise tolerance at high altitude.

The low level of oxygenation in the mixed venous blood was remarkable. During exercises near maximum effort, at barometric pressures of 347 and below, the mixed venous PO_2

was always between 10 and 14 mmHg (22), values lower than those reported from the coronary sinus at 3100m (9). One expects the coronary sinus PO_2 in our subjects to be as low or lower than the mixed venous blood. If both rate and contractile functions of the heart were maintained, then the heart must have been able, not only to tolerate, but also to function well, under extraordinary hypoxic conditions. Presumably, if the normal heart can adapt to such hypoxia, then similar mechanisms may be available in heart disease where the oxygen supply is limited.

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Legends

Figure 1. Relation of arterial oxygen saturation (SaO2) by ear oximeter to that measured directly in simultaneously sampled arterial blood. Shown (broken line) is the line of identity. The regression equation, the correlation coefficient, and the number of measurements are indicated. The relationship is similar to that previously reported for 5 measurements in 3 subjects during the AMREE expedition (24)

Figure 2. Relationship of heart rate at rest and during exercise to oxygen uptake at the barometric pressures shown. The regression lines are from the mean values in Table 4.

Figure 3. The time course of heart rate (filled circles) and oxygen saturation by ear oximeter (open circles) during a given exercise at the barometric pressures indicated. Each point is the mean of the same 5 subjects. The exercise intensities were 120 watts for subjects 1, 4, and 6, and 60 watts for subjects 3 and 8. At P_B 240 mmHg, the exercise intensity was reduced to 90 watts for subject 6. The resting heart rates are those immediately prior to the exercise shown and thus may differ from those given in Table 5.

Figure 4. Relation of maximum oxygen uptake (filled circles

and unbroken line) to the simultaneously measured heart rate for 5 subjects having measurements made at the barometric pressures of 760, 464, 347, 289, and 240 mmHg. The broken line shown for comparison is the regression line for submaximal exercise from sea level for these 5 subjects.

Figure 5A, top left. Relationship of cardiac output by thermodilution to oxygen uptake (VO2)for the barometric pressures of 760, 347, 282, and 240 mmHg. Shown here and in Figures 5B,C,D, and 6A,B, for each barometric pressure, are group means where all members of the group performed comparable exercise intensities.

Figure 5B, top right. Relationship of cardiac output to heart rate.

Figure 5C, bottom left. Relationship of stroke volume (cardiac output/heart rate) to oxygen uptake.

top right.

Figure 5D, bottom right. Relationship of stroke volume to heart rate.

Figure 6. Relationship of stroke volume to ventricular filling pressures at various barometric pressures. The number of individuals is shown.

A. Relation of stroke volume to right atrial mean pressure (RAM).

B. Relationship of stroke volume to wedge pressure.

Figure 7. Comparison of stroke volumes and ventricular filling pressures during air and oxygen breathing for the barometric pressures 760, 347, 282, and 240 mmHg. Shown as filled circles are the measurements during air breathing and the arrows indicate the progression to progressively higher exercise intensities. For some exercises at reduced barometric pressures stroke volume increases with increasing exercise despite a decrease in filling pressure. Resting measurements at reduced barometric pressures the heaviest exercise at all barometric pressures were repeated during oxygen breathing and are shown as open circles. Shown are group means for all of the subjects indicated in parentheses.

Table 1. Shown from left right, in this and subsequent tables are, subject identification by number, exercise intensity in watts, oxygen uptake as measured (VO_2) and as a per cent of the maximal value ($\%VO_2max$), heart rate (HR), stroke volume (SV), right atrial mean pressure (RAM), mean pulmonary wedge pressure (Wedge), pulmonary arterial mean pressure (PAM), and systemic arterial mean pressure (SAM).

Table 2. Notations are as in Table 1. In this and subsequent tables, SaO_2 is by IL Co-oximeter, Model 282.

Table 1. Hemodynamic measurements at sea level: 760 mmHg

Subject	Watts	VO2 (ml/min)	%VO2max (%)	HR	SV (ml)	RAM (mmHg)	Wedge (mmHg)	PAM (mmHg)	SAM (mmHg)
1	0	410	10	69	109	2	6	14	95
3	0	290	9	63	68	5	9	14	97
4	0	277	6	57	111	1	5	16	84
5	0	390	11	62	95	1	4	13	95
6	0	388	9	51	141	0	8	19	105
7	0	305	7	63	129	3	4	12	90
8	0	408	9	60	112	5	11	18	110
9	0	410	13	85	93	4	8	14	95
Mean	0	360	9	64	107	2.6	6.9	15	96
SEM	0	20	1	4	8	0.7	0.9	1	3
1	60	1390	33	106	134		9	23	120
3	60	1100	33	105	84	4	11	16	106
4	60	1130	26	72	179	4	10	22	90
5	60	1210	34	108	143	3	9	21	100
6	60	1430	34	87	187	11	18	27	110
7	60	970	21	78	188	6	10	17	92
8	60	1210	27	94	151	8	20	28	118
9	60	1186	37	111	142	3	10	16	95
Mean	60	1203	31	95	151	5.6	12.1	21	104
SEM	0	52	2	5	12	1.1	1.5	2	4
1	120	2030	48	128	160	4		22	110
3	120	1870	55	147	99	4	11	20	104
4	120	1640	38	93	220	4	11	24	90
5	120	1850	52	153	117	4	9	21	100
6	120	1826	43	96	189	14	22	30	105
7	120	1632	35	105	98	5		21	90
8	120	1770	40	109	168	10	20	30	144
9	120	1750	55	129	153	1	7	15	95
Mean	120	1796	46	120	151	5.8	13.3	23	105
SEM	0	46	3	8	15	1.5	2.5	2	6
1	180	2640	63	153	146		22	35	104
3	150	2190	64	159	87	5	11	21	102
4	180	2520	58	117	212	5	11	27	96
5	150	2190	62	159	126	3	9	21	100
6	180	2448	58	114	211	14	23	32	105
7	180	2100	46	129	168	5	12	22	92
8	180	2506	56	133	181	8	21	31	125
9	150	2045	64	150	139	1	7	16	100
Mean	169	2329	59	139	159	5.9	14.5	26	103
SEM	5	81	2	6	15	1.6	2.3	2	3
1	240	3320	79	156	155		17	36	104
3	210	2800	83	186	103	4	11	25	122
4	240	3300	76	132	183	5	11	27	90
5	210	2780	78	177	144	5	15	23	116
6	240	3148	75	132	196	16	26	39	118
7	240	2980	65	156	153	4	10	22	92
8	300	3555	79	162	185	12	30	42	142
9	210	2823	88	177	139	2	11	20	112
Mean	236	3088	78	160	157	6.9	16.4	29	112
SEM	11	102	2	7	11	1.9	2.7	3	6

Table 2. Hemodynamic measurements 6100m; PB = 347 mmHg

Subject	Watts	VO2 (l)	%VO2max (l)	HR	SV (l)	RAM (mmHg)	Wedge (mmHg)	PAM (mmHg)	SAM (mmHg)	SaO2 (%)
1	0	425	19	100	60	1	8	26	96	75
3	0	233	11	96	68	0	5	27	100	64
4	0	233	10	69						75
5	0	306	17	93	69	0	9	25	82	76
6	0	318	13	76	72	1	9	20	100	80
8	0	330	16	69	100	1	7	30	105	76
9	0	300	19	102	60	0	6	18	90	83
Mean	0	306	15	86	72	0.5	7.3	24	96	76
SEM	0	25	1	6	6	0.2	0.7	2	3	2
1	60	985	45	122	107	0	6	32	102	69
3	60	807	39	132	77	1	5	32	93	66
4	60	882	40	90						70
5	60	955	54	130	104	0	3	32	95	60
6	60	1080	44	84	136	5	10	38	110	76
8	60	963	45	105	139	2	7	41	120	63
9	30	725	45	111	91	1	4	24	92	69
Mean	56	914	45	111	109	1.5	5.8	33	102	68
SEM	5	45	2	7	10	0.8	1	2	5	2
1	120	1644	75	142	135	1	8	35	107	70
3	120	1288	63	159	97	3	8	36	106	65
4	120	1347	60	108						69
5	120	1368	77	146	114	3	6	39	91	58
6	120	1760	72	99	166	6	12	45	118	68
8	120	1564	74	124	149	3	8	48	120	56
9	90	1190	74	129	124	1	5	30	84	60
Mean	116	1452	71	130	131	2.8	7.8	39	104	64
SEM	4	78	2	8	10	0.7	1	3	6	2
1	180	2006	92	148	164	7	10	40	117	61
4	210	1936	87	128						67
6	210	2330	95	132	173	9	16	50	128	65
8	180	2120	100	138	180	6	12	53	128	55
Mean	195	2098	94	137	172	7.3	12.7	48	124	62
SEM	9	86	3	4	5	0.9	1.8	4	4	3

Table 3. Hemodynamic measurements 7620m; PB = 282 mmHg 8840m; PB = 240 mmHg

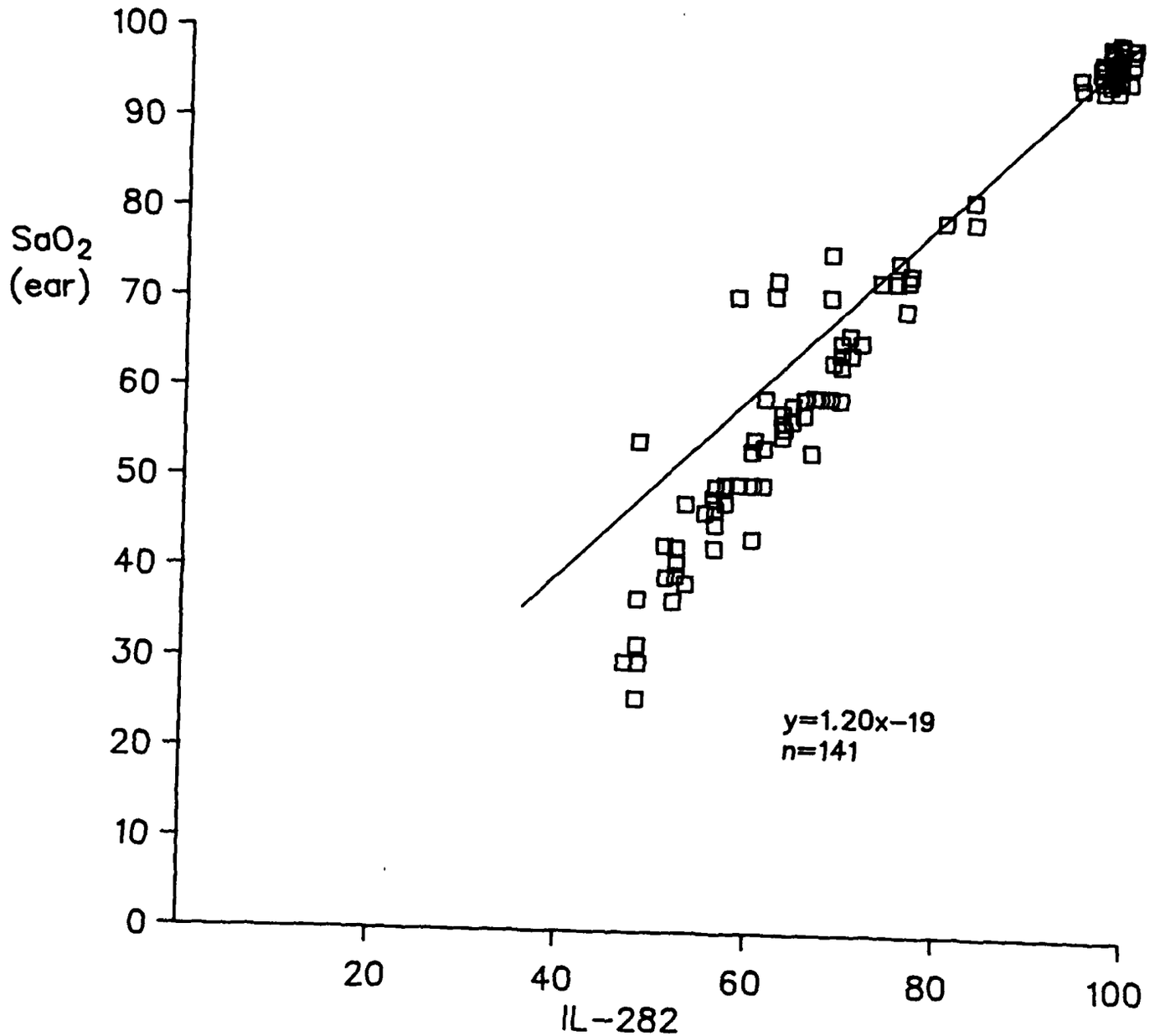
Subject	Watts	VO2 (ml)	%VO2max (ml)	HR	SV (ml)	RAM (mmHg)	Wedge (mmHg)	PAM (mmHg)	SAM (mmHg)	SaO2 (%)
PB=282 mmHg										
1	0	445	22	118	45	2	12	39	88	71
3	0	360	20	94	62	-1	4	26	84	68
4	0	435	24	88	85	0	6	41	96	68
6	0	410	22	91	53	0	9	30	88	73
8	0	381	21	82	98	-1	3	32	94	60
Mean	0	406	22	95	69	0	6.8	34	90	68
SEM	0	16	1	6	10	0.5	1.7	3	2	2
1	60	960	48	120	83	5		46	100	63
3	60	870	49	116	81	0	5	36	85	63
4	60	1050	57	111	124	3		55	102	62
6	60	900	48	93					105	69
8	60	1004	55	111	131	3	8	51	102	52
Mean	60	957	51	110	105	2.8	6.5	47	99	62
SEM	0	33	2	5	13	1		4	4	3
1	120	1540	77	134	93	8		53	133	56
3	90	1300	74	138	101	1	6	51	96	61
4	120	1270	69	124	159	9		60	116	58
6	120	1540	82	108					120	61
8	90	1575	87	111	159	3	4	51	116	(33)
Mean	108	1445	78	123	128	5.2	5	54	116	59
SEM	7	66	3	6	18	1.9		2	6	1
PB=240 mmHg										
1	0	370	36	118	64	3	10	37	90	56
3	0	360	38	102	59	0	3	25	80	57
4	0	415	33	93	99	1	7	38	76	62
6	0	350	25	80					122	66
8	0	435	35	103	103	2	10	33	112	56
Mean	0	386	33	99	81	1.5	7.5	33	96	59
SEM	0	17	2	6	11	0.6	0.7	3	9	2
1	60	810	79	120	95	7		48	115	51
3	60	870	91	130	82	1	6	37	100	48
4	60	970	78	120	143	2	9	55	96	52
6	60	960	70	108					140	57
8	60	1026	82	116	123	4	8	46	108	47
Mean	60	927	80	119	111	3.5	7.7	47	112	51
SEM	0	39	3	4	14	1.3	0.9	4	8	2
1	90	1000	98	124	124	8		52	115	48
4	90	1200	97	120	150			55	108	52
6	90	1290	93	111					150	53
Mean	90	1163	96	118	137			54	124	51
SEM	0	86	2	4					13	2
1	120	1010	99	122	131	9		52	120	30
4	120	1140	92	120	197			55	104	43
6	120	1380	100	111					160	37
Mean	120	1177	97	118	164			54	128	37
SEM	0	108	3	3					17	4

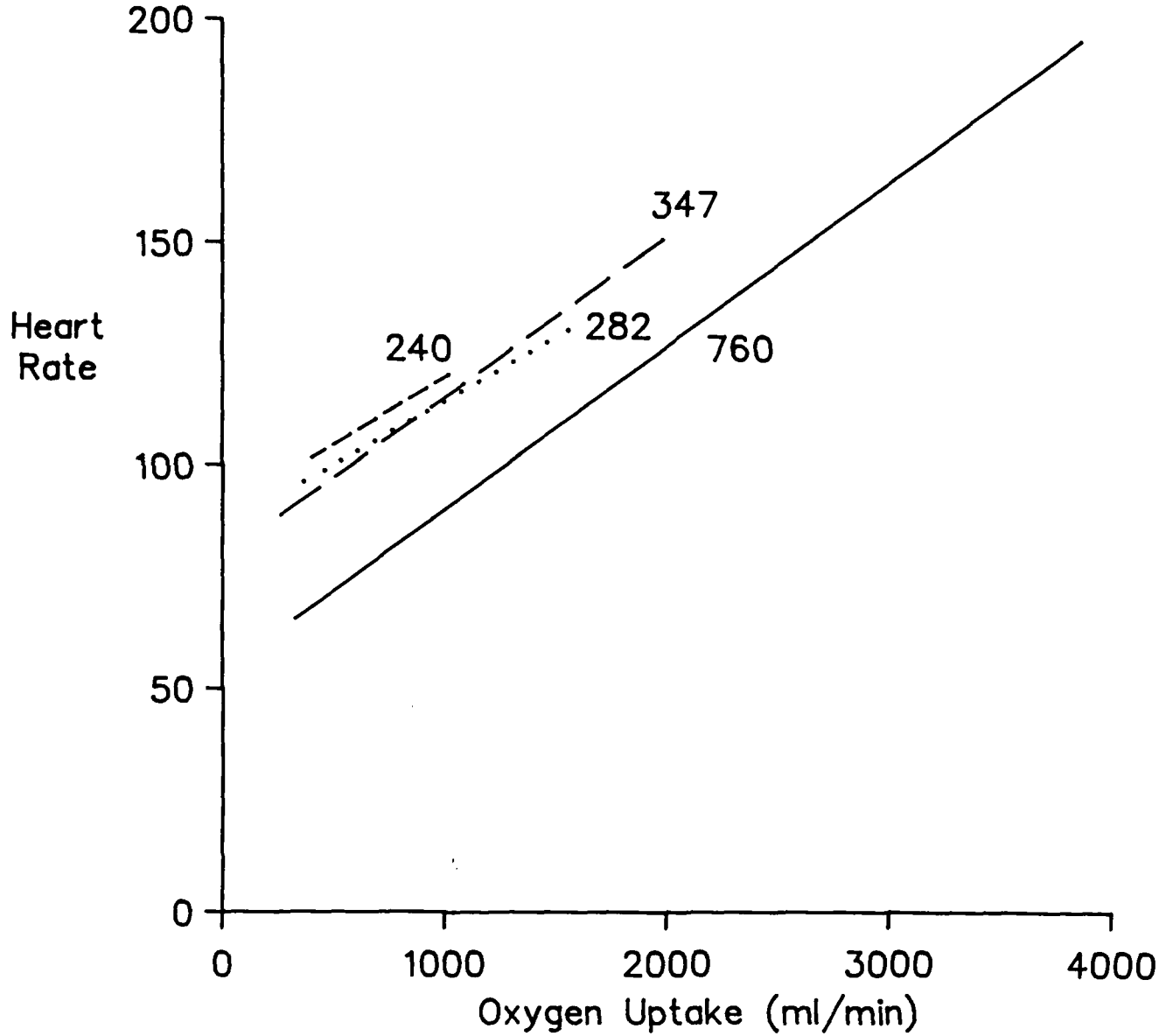
Table 4. Regression equations relating oxygen uptake (X) to heart rate (Y)

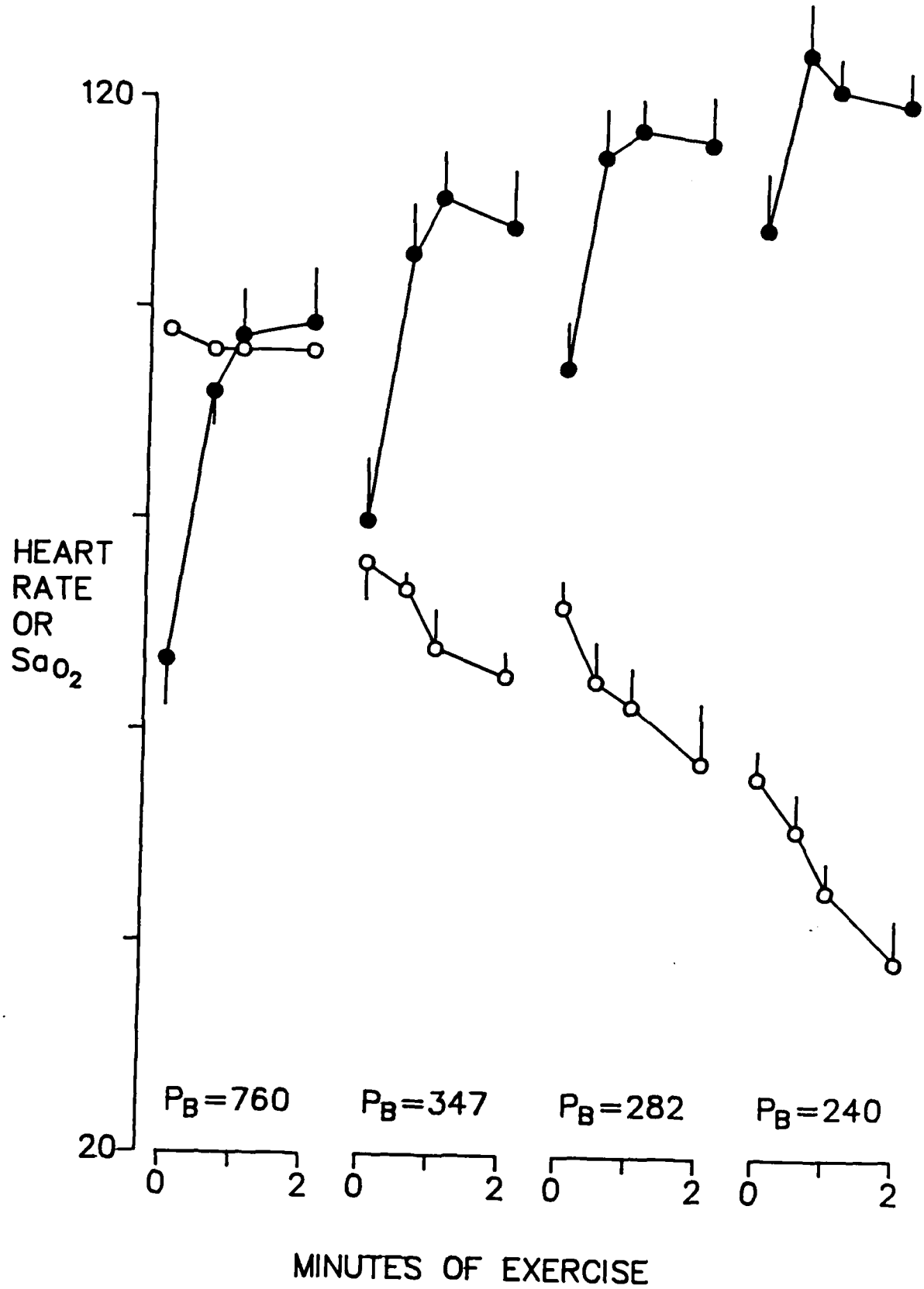
	a, slope (ml/beat)				b, intercept (beats/min)			
	PB=760	PB=347	PB=282	PB=240	PB=760	PB=347	PB=282	PB=240
Subject								
1	0.032	0.031	0.015	0.008	60	89	109	115
3	0.050	0.052	0.047	0.055	50	89	77	82
4	0.027	0.035	0.042	0.036	46	60	69	79
5	0.044	0.051			59	79		
6	0.031	0.027	0.015	0.031	40	61	83	72
7	0.038				47			
8	0.032	0.038	0.025	0.022	51	62	77	93
9	0.024	0.021	0.018		59	96	99	
Mean	0.037	0.036	0.027	0.03	52	77	86	88
SEM	0.003	0.004	0.006	0.008	3	6	6	8

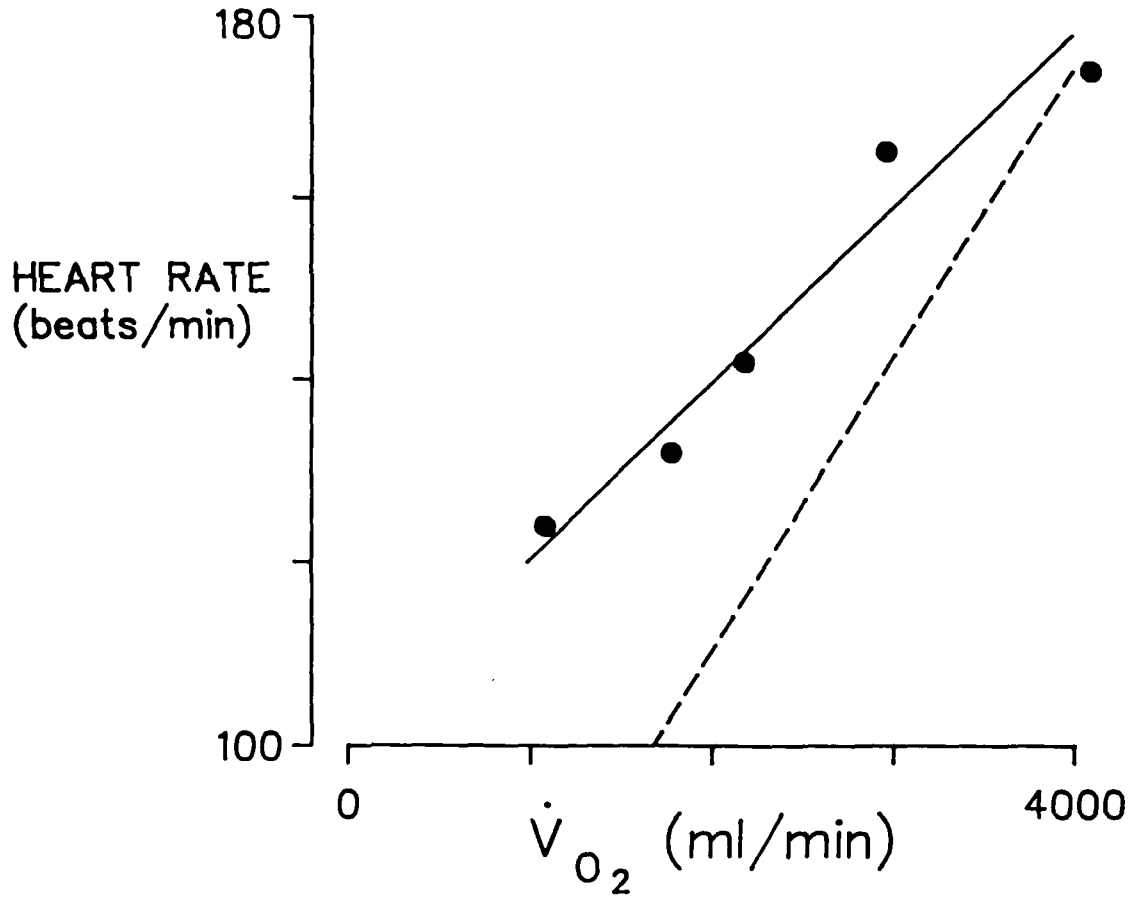
Table 5. Effects of oxygen breathing on heart rate and stroke volume

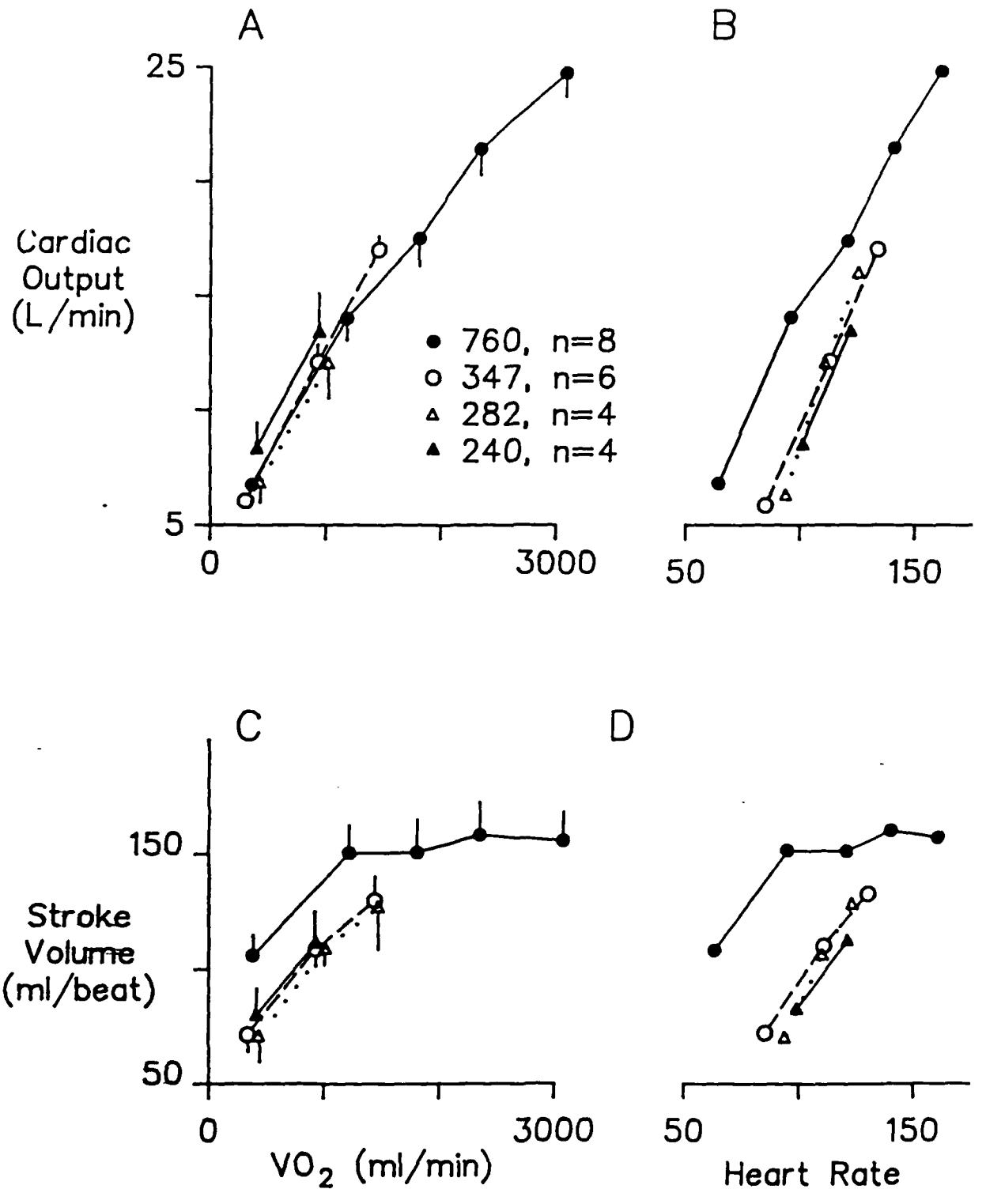
Subject	Resting measurements				Exercising measurements				
	HRair	HR O2	SVair (ml)	SV O2 (ml)	Watts	HRair	HR O2	SVair (ml)	SV O2 (ml)
				PB=760					
1	69		109		300	167	172		
3	63		68		210	186	192	103	80
4	57		111		360	162	162	222	201
5	62		95		210	177	171	144	132
6	51		142		360	162	168	207	203
7	63		129		360	189	168	148	186
8	60		112		300	162		185	
9	85		93		210	177	180	139	147
Mean	64		107		287	173	176	164	158
SEM	4		8		28	4	5	16	20
				PB=347					
1	100	90	60	52	180	148	144	164	113
3	96	108	68	43	120	159	138	97	85
4	69				210	128	92		
5	93	76	69	71	120	146	134	114	97
6	76	56	72	84	210	132	136	173	135
8	69	74	100	77	180	138	122	180	153
9	102	84	60	58	90	129	111	124	116
Mean	86	81	72	64	159	140	125	142	117
SEM	6	7	6	6	18	4	7	14	10
				PB=282					
1	118	107	45	37	120	134	134	93	93
3	94	88	62	55	90	138	128	101	72
4	88	90	85	47	120	124	112	159	138
6	91	84	53	58	120	108	100		
8	82	71	100	75	90	111	107	159	105
Mean	95	88	69	54	108	123	116	128	102
SEM	6	6	10	6	7	6	6	18	14
				PB=240					
1	118	102	64	58	120	122			
3	102	90	59	57	60	130			
4	93	82	99	72	120	120			
6	80				120	111			
8	103	61	103	121	60	116	114	123	101
Mean	99	90	81	77	96	120			
SEM	6	8	11	15	15	3			

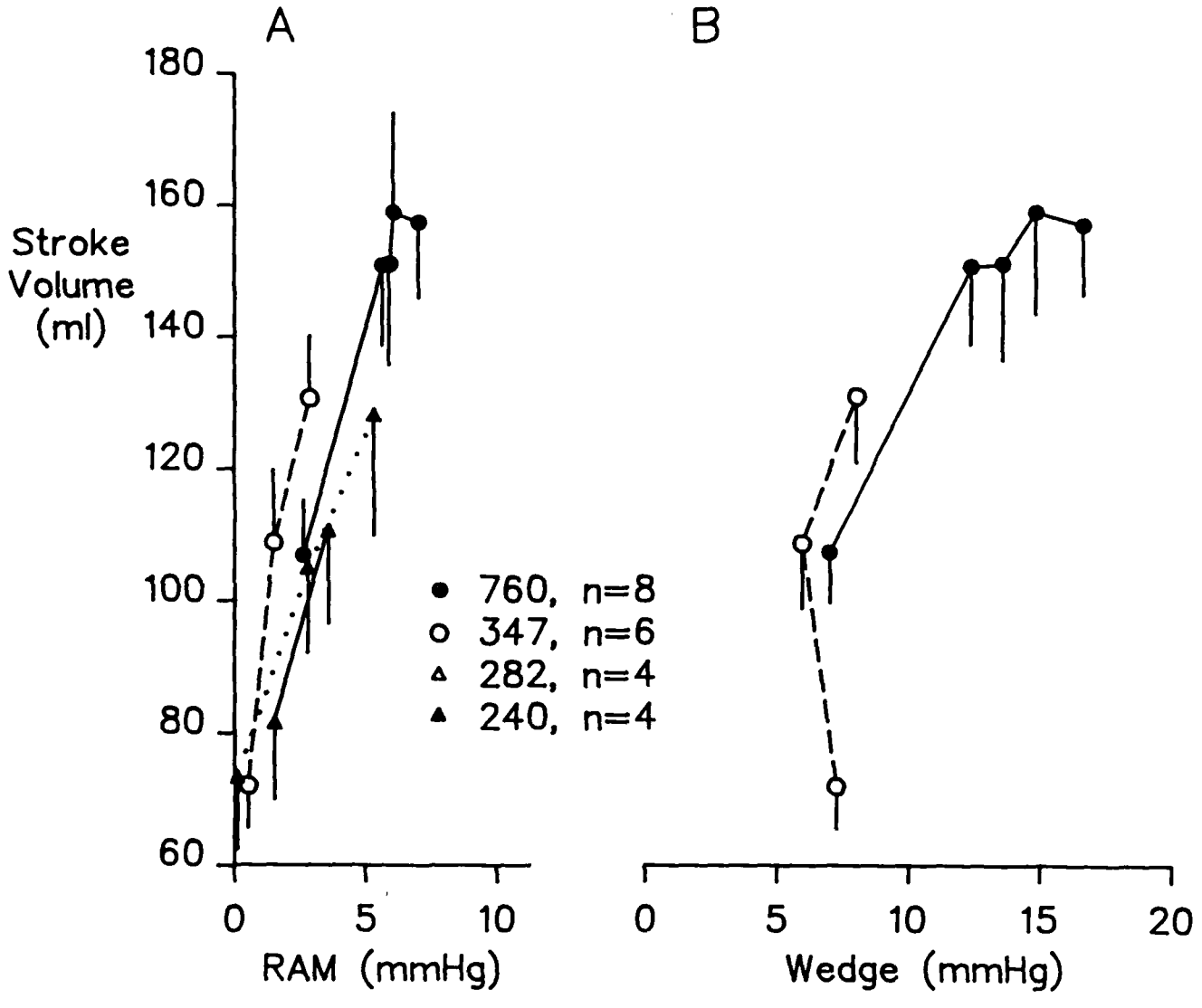

100
90
80
70
60
50
40
30
20
10
0
SaO2
(ear)
20
40
60
80
100
IL-282
y=1.20x-19
n=141

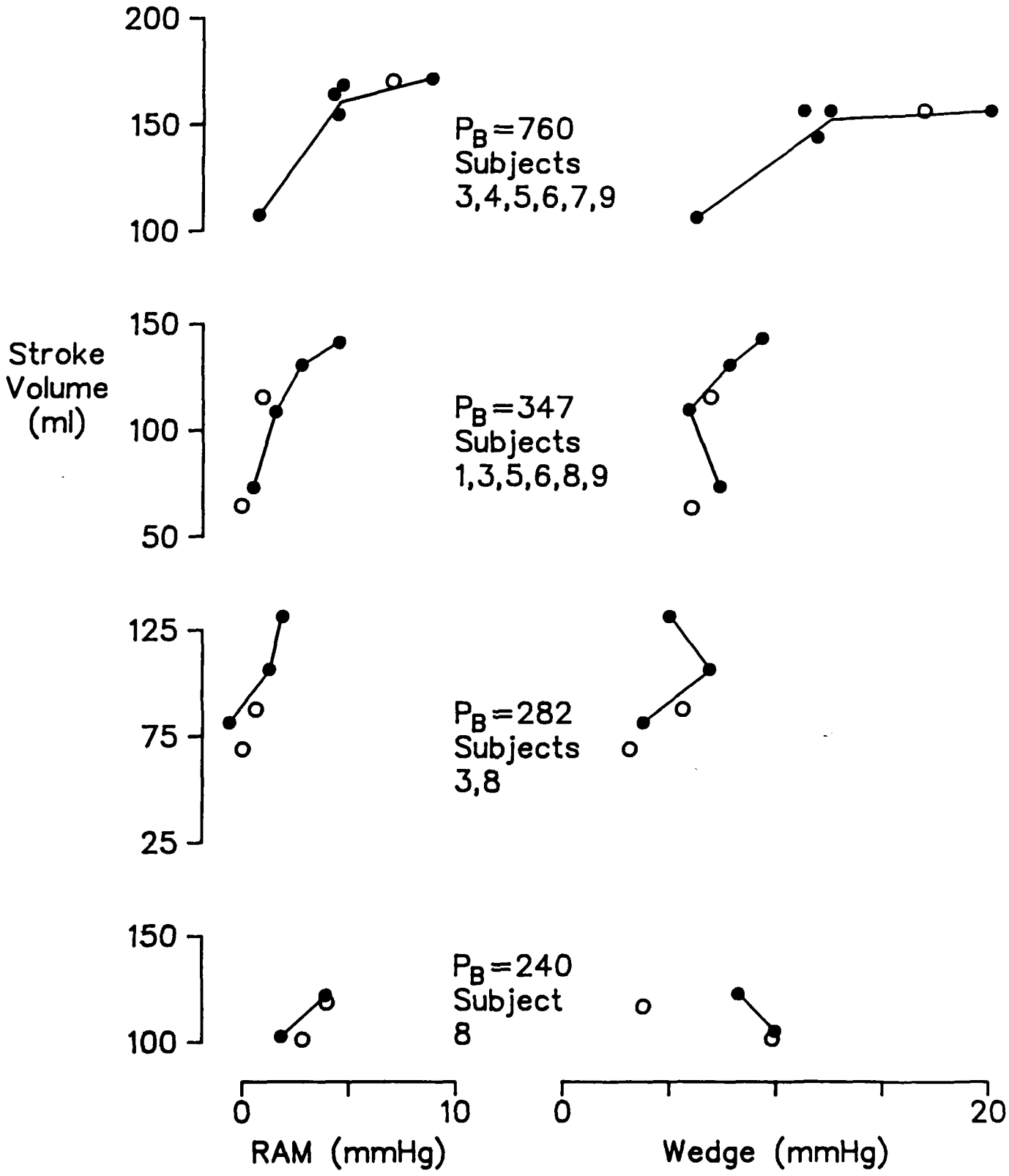

200
150
100
50
0
Heart Rate
347
282
240
760
0
1000
2000
3000
4000
Oxygen Uptake (ml/min)


120
20
HEART
RATE
OR
Sa_{O_2}
P_B=760
P_B=347
P_B=282
P_B=240
0
2
0
2
0
2
0
2
MINUTES OF EXERCISE


180
HEART RATE
(beats/min)
100
0
4000
$\dot{V}_{O_2}$ (ml/min)


A
B
C
D
25
5
Cardiac Output (L/min)
760, n=8
347, n=6
282, n=4
240, n=4
0
3000
50
150
150
50
Stroke Volume (ml/beat)
0
3000
VO_2 (ml/min)
50
150
Heart Rate


A
B
180
160
140
120
100
80
60
Stroke Volume (ml)
760, n=8
347, n=6
282, n=4
240, n=4
0
5
10
RAM (mmHg)
0
5
10
15
20
Wedge (mmHg)


200
150
100
P_B=760
Subjects
3,4,5,6,7,9
150
100
50
Stroke
Volume
(ml)
P_B=347
Subjects
1,3,5,6,8,9
125
75
25
P_B=282
Subjects
3,8
150
100
P_B=240
Subject
8
0
10
RAM (mmHg)
0
20
Wedge (mmHg)

END

11-86

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